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POLYBROMINATED DIPHENYL ETHER (PBDE) CONCENTRATIONS DECREASE WITH AGE: ANALYSIS OF POOLED HUMAN BLOOD SERUM IN THE AUSTRALIAN POPULATION

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Introduction

Polybrominated diphenyl ethers (PBDEs) are considered to be a cost effective and efficient way to reduce the possibility of product ignition and inhibit the spread of fire, thereby limiting harm caused by fires. PBDEs are incorporated into a wide variety of manufactured products and are now considered an ubiquitous contaminant found worldwide in biological and environmental samples[1].

In comparison to "traditional" persistent organic pollutants (POPs), the exposure modes of PBDEs in humans are less well defined, although dietary sources, inhalation (air/particulate matter) and dust ingestion have been reported [2-4]. Limited investigations of population specific factors such as age or gender and PBDE concentrations report: no conclusive correlation by age in adults [5, 6]; higher concentrations in children [7, 8]; similar concentrations in maternal and cord blood [9]; and no gender differences [6, 10].

After preliminary findings of higher PBDE concentrations in children than in adults in Australia[11] we sought to investigate at what age the PBDE concentrations peaked in an effort to focus exposure studies. This investigation involved the collection of blood samples from young age groups and the development of a simple model to predict PBDE concentrations by age in Australia.

Materials and Methods

Human blood sera was collected and pooled for analysis in 2002/03, 2004/05 and 2006/07. De-identified serum samples were obtained from Sullivan and Nicolaides Pathology from surplus stored sera that had been collected as part of routine pathology testing.

Prior to pooling, all samples were stratified according to age and gender. For the 2002/03 samples the age groups were: <16; 16 to 30; 31 to 45; 46 to 60 and >60 years. For the 2004/05 samples the age groups were: 0-4; 5-15; 16 to 30; 31 to 45; 46 to 60 and >60 years. For the 2006/07 samples the age groups were: cord blood; 0-6; 6-12 months; 1-1 ½; 1 ½-2; 2-2 ½; 2 ½-3; 3-3 ½; 3 ½-4; 4-6; 6-9; 9-12; 12-15; 16-30; 31-45; 46-60 and >60 years. Sera were collected from both males and females. Overall, 10552 individual samples were used to form 169 pools. For the 2002/03, 2004/05 and 2006/07 samples, 5729, 2403, and 2420 individual serum samples were used in 61, 24 and 84 pools, respectively.

The 2002/03 and 2004/05 samples were analysed at ERGO/Eurofins, Hamburg, Germany while the 2006/07 samples were analysed at CDC, Atlanta, USA. The following congeners were targeted for analysis by means of isotope dilution technique using HRGC/HRMS (high resolution gas chromatography/high resolution mass spectrometry) at

both laboratories: BDE-17, -28, -47, -66,, -85, -99, -100,-153, -154, and -183. The methodologies have been described previously[11, 12].

We developed a simple model with the aim of evaluating whether or not primary known exposure pathways (food, air, dust, breast milk) and clearance (half-life) data could be used to predict the measured age distribution and the peak age of maximum PBDE concentrations in the Australian population. The following assumptions were made: PBDEs are stored exclusively in body lipids; maternal concentrations equal newborn concentrations [9, 13]; and all PBDEs ingested via human milk, food, air and dust were absorbed and retained in the body. An infant was assumed to consume either breast milk or formula from 0 - 12 months with the addition of food from 6 months until breast milk/ formula was ceased at 12 months and food alone was consumed. The model was resolved on a monthly (30 day) basis for the first 24 months and then on a 3 monthly (90 day) basis for the next 24 months, then a yearly (365 day) basis from 4 to 65 years. The model was constructed in Microsoft Excel.

Concentrations of PBDEs in breast milk, dust and air were derived from our own studies[14, 15]. Food and formula data were calculated from a report on PBDEs in food in Australia[16]. Breast milk intake was based on 778 g/day for the first six months followed by a 10% decrease each month until cessation at 12 months. Data presented in this paper are for BDE-47.

Ethics approval for this study was granted on 20 September 2002 by The University of Queensland Medical Research Ethics Committee.

Results and discussion

Overall, ΣPBDEs were detected in all pools of human blood serum ranging from 4.2 to 108 $ng.g^{-1}$ lipid. A trend by age was observed in samples from all three collection periods. The 2006/07 samples focused on the younger age groups and therefore the age trend was more apparent with the highest ΣPBDE concentration (108 $ng.g^{-1}$ lipid) detected in a pool of serum from 2 ½ - 3 year olds. ΣPBDE concentrations for the 1-5 year olds were around twice those of cord blood, < 1 years, 6-12 years and 13-30 years and around four times those of the > 31 year olds (Table 1).

Table 1. Mean ± standard deviation ΣPBDE concentrations ($ng.g^{-1}$ lipid) by age (years) for 2006/07

Age (years)	ΣPBDE concentration (ng/g lipid)
cord blood	24.1± 14
<1	23.9 ± 14.6
1-5	41.9 ± 15.5
6-12	25.8 ± 9.3
13-30	20.6 ± 16.8
>31	9.7 ±2.4

No relationship was found between PBDE concentrations and gender. No temporal trends were apparent in PBDE concentrations between the collection periods. The congener profile of PBDEs in the 2002/03, 2004/05 and 2006/07 samples was dominated by BDE-47. No differences in congener profile by age or gender were observed.

Using the model we predicted PBDE concentrations in the Australian population by age and gender taking into consideration consumption of breast or formula milk in infancy for BDE-47. The modeled data were compared to the measured data by year of collection. As there was no difference in PBDE concentrations by gender, data for both males and females are included and the modeled data (breast fed infants) is the mean of the predicted concentrations for males and females (Figure 1).

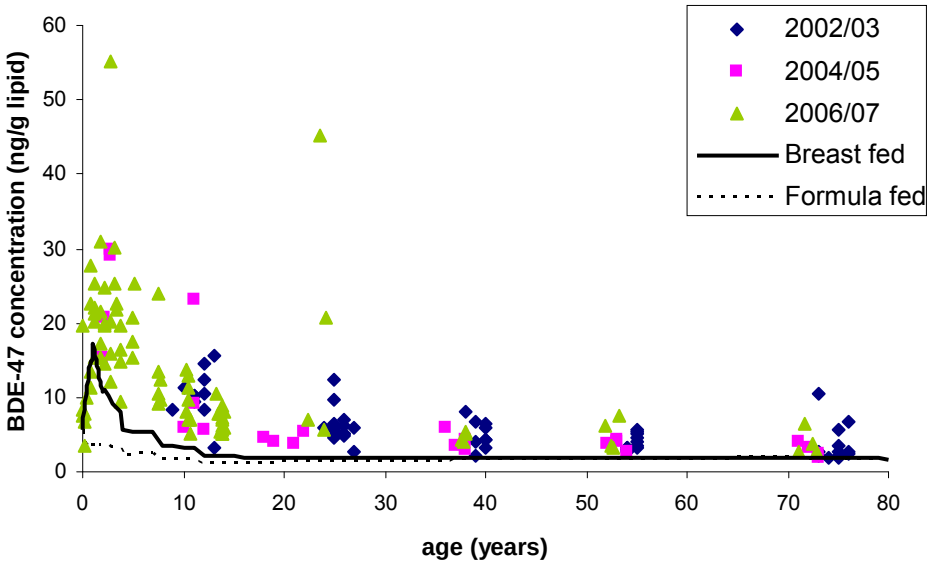

Figure 1 ΣPBDE concentration (ng.g^{-1} lipid) in each pool by the mean age (years) of donors in each pool for 2002/03, 2004/05, 2006/07 and the predicted data (breast and formula fed) generated by the model

Predicted concentrations peaked at age 13 months for breast fed infants however predicted concentrations for formula fed infants never exceeded the initial birth concentration. The model predicted the trend in PBDE concentration observed in the measured data, however the predicted concentrations underestimated the measured data from the Australian population. The differences in measured and predicted data indicate that either intake sources other than breast milk, food, air and dust contribute to the human concentrations of PBDEs or that the half-lives have been underestimated. The model most accurately predicted concentrations measured in the > 36 years age groups indicating that known input and clearance data were more reflective of exposure. The underestimation of concentrations in young people may indicate that input and clearance data are not yet fully understood for this age group. While breast fed infants exceed formula fed infants other potential sources in the young age group, not accounted for in the model, include increased exposure from mouthing and sucking products either potentially treated with PBDEs or covered in dust contaminated with PBDEs from the indoor environment and exposure to child-specific products such as car seats, prams, mattresses and toys which may also be a source of PBDEs. In addition, overall body burden of PBDEs may be determined by individual metabolic differences that affect rates of retention/ sequestration [17] and these factors were not accounted for by the model.

The study provides important information on the concentration of PBDEs in over 10000 samples across the lifespan from birth to greater than 60 years. The peak in concentration in young children indicates that investigation of the missing sources and exposure pathways as well as metabolic factors which may be influencing the body burden of these chemicals in the young age groups is required.

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